



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

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MEMORANDUM

SUBJECT: Update to Review of Azinphosmethyl Incident Reports,
DP Barcode 256164, Chemical #058001, Rereg. Case #0235

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Since the review of azinphosmethyl incident reports dated August 21, 1997 (DP Barcode 238115) some additional pertinent information on Poison Control Center exposures and cholinesterase monitoring data has been found. The earlier review reported 39 occupational and 76 non-occupational symptomatic cases due to exposure to azinphosmethyl (as a single exposure rather than exposure to multiple products) between 1985 and 1992. An additional four years of data covering 1993-1996 found another 14 occupational and 49 non-occupational symptomatic cases due to azinphosmethyl. Overall there does not appear to be any trend from the earlier years to the 1993-1996 time period, though a decline in occupational cases and an increase in non-occupational cases is suggested. However, trends in Poison Control Center data can be affected by the changes in participation by individual centers over the years. Typically, non-occupational exposures occur when bystanders are exposed to field residue or spray drift.

Additional information has been obtained concerning exposures to azinphosmethyl that included measurements of blood cholinesterase levels. This information is summarized below.

California accessed medical monitoring records for 542 agricultural pesticide applicators under medical supervision in

1985 for exposure to the more toxic cholinesterase-inhibiting organophosphate and carbamate pesticides (Ames et al. 1987, 1989). In California, cholinesterase monitoring is required for all pesticide applicators who handle Toxicity Category I or II organophosphate or carbamate pesticides for 30 hours or more in any 30 day period. To be included in the survey, the worker had to have at least one pre-exposure (baseline) cholinesterase measurement and at least one exposure value (mid-season). A data-call-in was issued by the California Department of Food and Agriculture and local Agricultural Commissioners through pesticide application firms to their medical supervisors. Follow up letters were sent and phone calls made to employers, physicians, and laboratories performing tests, but significant under reporting is likely to have occurred. Therefore, these workers may not be representative of all workers undergoing medical monitoring in California. However, they do represent exposure effects verified by medical laboratories. Cholinesterase activity depression of 20 percent or more below baseline was observed in 127 or 23 percent of the 542 workers. Depression of 20 percent or more below baseline represents strong evidence of exposure (Gallo and Lawryk 1991).

Specific pesticide exposure was available for 94 of the 127 cases, based on usage records for the previous two weeks. Of these, 31 percent had been exposed to mevinphos, 21 percent to methomyl, and 21 percent to parathion, the three leading pesticides responsible for cholinesterase inhibition. Of the 94 cases with inhibition, 11% had exposure in the past two weeks to azinphosmethyl. Note that many of the workers were exposed to two or more pesticides during the two weeks before they had cholinesterase depression of 20% or more. Twelve of the workers in this study were reported to have pesticide-related illnesses by their physicians. These data demonstrate that agricultural workers, who mix, load and apply the more toxic pesticides are subject to significant levels of exposure despite the considerable restrictions in place to prevent exposure.

California has maintained a Pesticide Illness Surveillance Program with consistent data collection procedures since 1982 (Data tabulations provided by Louise Mehler, M.D., California EPA). From 1982 through 1996 there were 63 illnesses (with a possible, probable or definite relationship) that included taking a cholinesterase value and exposure to azinphosmethyl. In 12 of these cases, azinphosmethyl was considered the primary pesticide responsible for poisoning. Of the 63 cases with some exposure to azinphosmethyl, 22 (35%) had below normal levels of cholinesterase or evidence of a marked increase in cholinesterase (20% or more) subsequent to their exposure. Of the 12 cases where azinphosmethyl was determined to be the primary cause of poisoning, five (42%) had

evidence of cholinesterase depression. The evidence consisted of cholinesterase depression below laboratory normal values in three of the five cases and subsequent increases in cholinesterase of 40% or more reported in two of the five.

A study of 20 California peach harvest workers was conducted to test different biomarkers of exposure (McCurdy et al. 1994). Cholinesterase measurements were taken 6 days prior to exposure, on the third day of exposure, and 44 days after initial exposure. Thirty days prior to exposure, azinphosmethyl had been applied to study orchards at a rate of 1.5 pounds per acre. The re-entry period for azinphosmethyl in California is 14 days. In comparison with baseline median values, red blood cell cholinesterase values decreased 7% after 3 days of exposure and 19% over the 6-week harvesting season. The higher reduction in cholinesterase at the end of the study rather than on day 3 of exposure was unexpected and thought to be due to an improper handling of samples collected on day 3. This study did not examine health outcomes in the workers.

A similar study of peach harvesters in California was reported by Schneider et al. 1994. In this study 23 harvesters (exposed) and 10 sorters (considered to have minimal exposure) had baseline cholinesterase levels taken and then entered an orchard 51 days after an application of 1.5 pound per acre of azinphosmethyl. The reduction in plasma cholinesterase was not significant when harvesters were compared to sorters. However, red blood cell cholinesterase values for harvesters were significantly below those of sorters for two post-exposure blood draws as measured by three testing methods. Compared to their baseline levels exposed harvesters experienced a 10-20% decline in red blood cell cholinesterase. No symptoms of organophosphate poisoning were reported by any of the workers.

Two studies reported in the late 1970s also examined field workers exposed to azinphosmethyl in California. In a study reported by Kraus et al. (1977) 21 peach thinners were monitored who entered the orchard 12-18 hours after spraying. A 15% decline of whole blood cholinesterase was reported over the five days of the study. There were no clinical signs of organophosphate poisoning. Richards et al. (1978) reported on a similar study of peach thinners. In this study eight workers were exposed thinning peaches in a field treated with azinphosmethyl and experienced a 8% decline in red blood cell cholinesterase. No workers reported signs of organophosphate poisoning.

Conclusion

Field workers exposed to residues of azinphosmethyl may

experience significant declines in red blood cell cholinesterase. In the monitoring studies examined for this review none of the workers reported ill effects that could be directly attributed to cholinesterase inhibition. Poison Control Centers continue to report symptomatic cases due to azinphosmethyl at a rate of about 16 cases per year.

References

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cc: correspondence
azinphosmethyl file (chemical no. 058001)